especially when an accompaniment of acute arthritis, treatment of the infection and local applications to the precordia will often suffice. When the progress is unsatisfactory, the physical signs increasing, and the diagnosis doubtful, paracentesis is imperative.

7. Frequent examinations during the infection and for some time after are very important. A thorough search for and the eradication of foci should be conducted in every case.

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A TREATMENT OF GASTRIC ULCER BASED UPON ESTABLISHED CLINICAL, HISTOPATHOLOGICAL AND PHYSIOLOGICAL FACTS.[1]

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Even a casual acquaintance with the literature of peptic ulcer demonstrates that treatment of the affection has been largely empirical. Various types of therapy appear to have obtained a vogue because their application either relieved symptoms or appeared to prevent a fatal issue. Success of a mode of treatment has been judged mainly from its effect upon the immediate condition and not with respect to the ultimate outcome of the affection. It is most unusual to observe statistics of hospitals or private practices that indicate the status of patients treated after intervals of five to twenty-five years. Hospital records generally state that a peptic ulcer patient has been discharged either "cured," "improved," "not improved," or has "died." With exception of the fatal cases, facts are usually not available regarding the future course of the ailment, inasmuch as these patients frequently seek the advice of another physician should their disability return after a long and expensive course of treatment at the hands of a former physician. It is also of interest to observe that of a half-dozen experts non-surgically treating peptic ulcer by radically different regimes the percentages of cure show a range of but five to ten points. We have also been impressed by the fact that at the most competent hands, certain ulcers resisted all types of treatment alike. It has seemed to us that unsatisfactory as the treatment of peptic ulcer might be,

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it were possible to devise a type of management that at least had the merit of resting upon certain basic principles. It likewise appears to us that methods of therapy are dependent upon information that has been returned by recent histological, physiological, and clinical investigations.

I. The Problem.

1. *Clinical.* Peptic ulcer is a disease which may or may not exhibit symptoms or signs. It is an ailment which, in its uncomplicated form, may be so closely mimicked clinically by non-ulcerous dyspepsias as to deceive the most experienced diagnostician. Until complications occur it is a disease characterized in 84 per cent. of instances by a peculiar "periodicity," and which often manifests itself by recovery and relapse frequently wholly independent of type or duration of treatment. It is associated with a form of gastric malfunction whose cessation does not generally indicate eradication of its exciting causes. It is a disease about which little prophecy respecting its extent or its future course can be made from its clinical symptomatology. Acuteness of symptoms does not wholly indicate the histopathological changes existing at the diseased foci—for acute manifestations histologically can be engrafted upon chronic inflammatory tissue pathologically. The most serious clinical outcome may frequently follow upon spontaneous or so-called therapeutic cure with the subsidence of the initial dyspeptic upset. Stenoses, gastric malformation, involvement of extragastric viscera, fatal hemorrhage, or malignant transformation may occur with comparatively slight clinical manifestation or certainly with relatively little change in the patient's complaints. Peptic ulcer is an ailment entirely indistinguishable clinically from the early appearance of gastric cancer, syphilis, or tuberculosis. It is a disease which but rarely occurs in individuals not already affected with other clinical abnormalities—infections about the head, throat, and neck; abdominal disease, *i. e.*, inflammatory changes in the appendix, gall-bladder, perigastric lymph nodes, pelvic structures, etc.; disturbances in the circulatory, eliminative, nervous, or lymphatic systems; and malfunctions of that group of glands concerned with food digestion and assimilation. Finally, it is an ailment of almost universal distribution, and one capable of producing such disturbance of the physical and mental health as to demand relief.

2. *Nature of Peptic Ulcer as Shown by Experimentation.* Before discussing the possibility of curing an ailment, it would appear quite essential that one be adequately informed regarding the character of the existing malfunction. That such inquiry is especially warranted with respect, gastric ulcer is indicated by the observation that scarcely a year has passed since the classic monograph of Virchow that has not seen a new attempt at explaining

the cause of ulcer of the stomach. Usually, closely attached to this experimental or clinical investigation there has also been cited a supposedly new method of healing the affection. That the majority of these contributions have proved unsatisfactory is clearly indicated by a recent elaboration of the "corrosion theory" of the origin of peptic ulcer, a theory pronounced inadequate forty years ago by von Leube, and often since revived.

Study of the affection demonstrates that a narrow conception of gastric ulcer is impossible. Competent observers have experimentally produced gastric ulcer by a wide variety of methods. It has been shown that gastric ulcers differing slightly in type histologically can be caused equally as readily from the systemic as from the local intragastric point of attack. Some of the more familiar causative agents are: (*a*) *Bacteria* (pneumonococci by Dienlafoy; staphylococci by Widal and Meskay; Staphylococcus aureus by Letulle; B. pyocyaneus by Charrin and Ruffer; B. dysenterii by Chantemesse and Widal; lactic acid bacillus and B. coli by Rodet and Zaidmann; streptococcus by Rosenow); (*b*) *Bacterial toxins* (pyemias by Lebert and by Cohn; diphtheria toxin by Enriquez and Hallion and Rosenau and Anderson); (*c*) *cutaneous burns* (Welty, Poufick, Silberman, Parascandolo); (*d*) *poisons of metabolic origin* (Bolton's "gastrotoxin" injected intraperitoneally or subcutaneously); (*e*) *extrinsic poisons* (mercury salts, arsenious acid, cantharidine, vegetable alkaloids, piloearpin and atropin, copper sulphate); (*f*) *poisons introduced into the stomach* (corrosives, caustics); (*g*) *alterations in the stomach's circulation* (vascular blocking, adrenaleetomy, thrombi, emboli, arterioselerosis or nervous inhibition, external pressure); (*h*) *artificiol pyloric spasm or stenoses* (Hamburger and Friedman); (*i*) mechanical trauma. Whatever may be the experimental mode of production, the types of ulcer resulting appear to vary but slightly. The lesions produced are essentially similar. There is inflammatory edema, loss of surface epithelium, hemorrhage, necrosis, glandular destruction, and frequently infection. In the healing of ulcers, irrespective of the way they may have been produced, the basic features consist in development of protective connective tissue by hyperplasia, with resultant scar.

The experiments of Bolton and of Rosenow are particularly interesting. The latter investigator has recently revived the bacterial theory of ulcer causation. By intravenous injections of streptococci usually obtained from throat and mouth lesions which have undergone a certain mutation culturally, Rosenow has succeeded in producing both acute and chronic gastric ulcer. From these ulcers he has been able to recover organisms, morphologically and culturally, similar to those with which, experimentally, he produced the lesions. He also has been able to demonstrate the inter-relationship of infections of the appendix, gall-bladder and

pancreas, and peptic ulcer with regard the streptococcus. He has shown the possibilities of organisms infecting the mouth, throat, or head sinuses gaining access to the general circulation and contributing to intra-abdominal pathology, or, by direct infection of the alimentary tract producing or aggravating ulceration. While Boetcher, Letulle, Dieulafoy and others many years ago advanced similar experimental proof, yet Rosenow, by tissue culture, has emphasized the significance of bacterial action in association with local or disseminated foci of chronic infection. However, as the definite causative factor of gastric ulcer, and particularly with respect its being *only cause*, Rosenow's bacterial proof cannot be wholly accepted. The ulcers which he has produced are not in any way dissimilar from those produced by other investigators using organisms other than the streptococcus or those ulcers caused by a wide variety of non-bacteriological agents. Rosenow has failed to show that the streptococci recoverable from injured mucosæ in the alimentary tracts of his experimental animals have not been secondarily deposited there as a consequence of some preceding, local, cellular fault. Control experiments using organisms other than streptococci are likewise lacking.

Antedating Rosenow's observations, and apparently being more basic in principle etiologically, are those of Bolton. This investigator has shown by an extensive series of experiments that *non-bacterial* tissue injections quite uniformly produce all grades of peptic ulcer in susceptible animals. Sterile emulsions of the gastric mucosa, of the appendix, gall-bladder, or liver injected intravenously or intraperitoneally produce in animals a toxic serum. At the end of from ten to fourteen days this serum has such selective affinity that its introduction into the circulation of animals produces necrosis and ulceration in the organs from which the original cell emulsions were made. The toxicity of the serum persists and may produce its effects after more than a year. The serum gives the various test-tube reactions for immune bodies, namely, lysins and precipitins. The most pronounced reactions are with gastric cell emulsions. Their injection rapidly causes acute hemorrhagic ulcers, which ulcers may later become chronic. Saturation of the serum with gastric cells before injection, robs the serum of its toxic qualities. Bolton has called this immune serum "gastrotoxin."

Bolton's gastrotoxic serum appears to indicate that, as result of a wide variety of ailments or systemic constitutional affections, disintegration of body cells may occur. The initial agents causing the malfunction form a great and dissimilar group. The poisons elaborated are of specific nature with respect the tissue from which they are derived. It would seem that the demands constantly being made upon the stomach result in a great cellular waste. The cellular waste satisfies the essentials of a protein poison. When

opportunity occurs this poison is capable of producing local cell changes in the gastric mucosa, which changes furnish the *anlage* for a subsequently developing ulcer. Once this initial local injury has been done to the gastric lining, the autodigestion of the mucosa by gastric juice or its ability to harbor bacteria are possible, and *not until then.*

The above brief summary indicates that we cannot expect to ever establish a single definite cause of gastric ulcer. The immediate causes of ulcers vary widely and are highly individual. The essential point is that through some malfunction a point of least resistance occurs in the gastric lining and *normal* gastric physiology is permitted to produce an *abnormal* tissue change. *It would likewise appear that we have no basis for regarding gastric ulcer as a distinct disease entity.* It seems more proper to consider that ulceration of the gastric mucosa is a local accident in association with a systemic upset, the nature of which upset may be extremely varied. When once the damage has been done locally in the stomach wall, whatever may be the primary cause, the future course of the process is similar. Pepsin and hydrochloric acid attack the inert gastric cells, necrosis results and ulcer is established. The course of the process depends mainly upon the persistence of the underlying systemic causes, variation in the quality of the gastric juice, local intragastric trauma (as by food, bacteria etc.), and the influence of the intrinsic muscles forming the stomach wall. Such facts strongly suggest that *peptic ulcer presents aspects indicating its being a self-limited disease.*

3. *Histopathological.* From the inception of symptoms clinically, it is impossible to prognosticate the future course of any peptic ulcer. While many ulcers destroy relatively little gastric mucosa and produce harmless scarification, thus evidencing a tendency to spontaneous cure, other ulcers, seemingly equally innocuous clinically, progress rapidly through various pathological stages and are very shortly associated with stenoses, extensive callous formation, hemorrhage, perforation, or malignancy. However, chronicity in the histopathological sense by no means indicates that a peptic ulcer is old in terms of months or years. Malignant change in ulcer edges may occur quickly and in no way point to the long existence of a previously benign affection. It is of much significance to observe variation in the appearance of pathological transformations in ulcers situated similarly in various parts of the stomach. While fully 85 per cent. of all gastric ulcers occur in the pylorus, antrum, and along the distal four-fifths of the lesser curvature, and while in these positions the greatest damage can be done by ulcer, yet just as striking changes occur in ulcers situated in other portions of the viscus. It is of importance to note that fully two-thirds of the gastric ulcers occur in the portion of the stomach whose mucous membrane is not devoted to the elaboration and secretion of hydro-

chloric acid. It would also seem to be of significance to recall that the majority of peptic ulcers occur in that part of the stomach in which the greatest circulatory, muscular, and nervous activity is manifest; that the fewest ulcers occur in that portion of the viscus which is fixed and which has to perform mainly the duties of a food receptacle and an acid-secreting organ.

4. *Physiological.* (*a*) *Chemical.* It is a striking commentary upon the empirical nature of standard treatments of peptic ulcer that they have as their basis the principle that gastric chemistry has been greviously upset. It has been supposed that this disarrangement has been caused either by the development of ulcer or that the upset itself has produced the ulcer. This chemical disturbance is generally put down as being in the nature of an overproduction of acid gastric juice and pepsin. It is presumed that the so-called "corrosive effects" of this overactive digestive juice produces gastric ulcer or prevents healing of one already present. It is significant to observe, in the first place, that the normal gastric juice frequently varies in strength well beyond the range of the so-called increases which are often demonstrable when gastric ulcer exists. In a recent study of 500 cases of peptic ulcer, in but 40 per cent. did I find a free HCl concentration above 0.3 per cent. In 35 per cent. the acidity was well within the "normal" range, and that in the remaining 25 per cent. the acidity was reduced or was entirely absent. Similar observations have been recorded experimentally by Carlson, and in man by Rehfuss and his coworkers. Moreover, the portions of the stomach which are least the site of gastric ulcer are those in which, physiologically, the concentration of hydrochloric acid is greatest and where the acid is in nascent form. That there are causes of gastric ulcer other than the variation in the hydrochloric acid concentration is also supported by the observation that in bile-free stomachs large amounts of hydrochloric acid in the concentration of as high as 0.6 per cent. mixed with pepsin may be placed in the stomach and yet not produce ulcer. It is also evident that the primary damage which results in gastric ulcer is not dependent wholly upon acid gastric juice by the ancient observation that if this were the case the stomach would digest itself. Clinical and histopathological evidence indicate that when once the initial damage to the gastric lining has been brought about from whatever cause, digestion of partly devitalized or necrotic mucous membrane may occur with equal readiness in the presence of normal or even subnormal gastric acidity. With these facts in mind it seems strange that treatment of gastric ulcer has largely rested upon a chemical foundation, which chemical foundation was extremely unstable and shifting. It has been presumed that in ulcer stomachs, overacidity was present, and that this overacidity must be counteracted by alkalinization before healing could take place. It has been presumed that the pain of gastric

ulcer existed as a consequence of irritation and "corrosion" of raw mucous surfaces by gastric juice rich in hydrochloric acid and pepsin even though the researches of Pawlow, Rehfuss, Carlson, Hertz, Hamburger and others have demonstrated that pain is not uniformly present when gastric acidity is highest, and that in known ulcer cases the introduction of high percentages of acid fails to produce pain. The relief from pain in gastric ulcer cases is quite as prompt when alkalies are administered in low acid cases as when such are used in high acid cases. Prompt relief of pain in such instances is also secured by the exhibition of lavage, alcohol, opiates, diet, etc. It would seem, therefore, that medical treatment of peptic ulcer based upon chemical upsets supposed to be associated with the ailment presents much that is empirical, irrational, and unscientific.

(*b*) *Physiological Motor Consideration.* Modern physiological investigations have been especially rich in elucidating the motor activity of the stomach under normal and pathological conditions. For this knowledge we are indebted to the development of the roentgen-ray and the tireless researches of Pawlow, Cannon and his pupils, and to Carlson. These investigators have established the significance of certain gastric motor cycles. They have shown that these cycles occur with almost mathematical precision. Upon these constants it would appear to be possible to base clinical observations and therapy. It would also seem that the mechanical factors concerned with digestion are of greater importance than are variations in the secretory function. It is with the mechanical features in digestion that we purpose to deal in suggesting a rational régime in the treatment of peptic ulcer. In order to emphasize the points mentioned below in detailing the type of treatment which we advance, the chief facts established by physiological investigation with regard the mechanical activity of the stomach now will be summarized:

The fasting stomach is in a state of tonic contraction. It is rarely empty. Its contents contain both hydrochloric acid and pepsin. These secretions apparently aid in preserving gastric tone, in digesting mucus, dead bacteria, and desquamated cell detritus. Hunger is manifested by rhythmic gastric systoles. These precede appetite desire and cause an unpleasantness that leads to eating. Repeated swallowing motions cause inhibition of gastric tonus. Eating starts gastric-juice secretion. Food entering the stomach initiates the peristaltic movements peculiar to gastric digestion. These peristaltic movements continue so long as food remains in the stomach. They pass from the pars media toward the pylorus in rhythmic sequence in a given case with equal intensity, thus maintaining a constant pressure in the antrum. The proximal third of the stomach acts mainly as a sac or reservoir and is comparatively free from peristaltic activity. The acid reaction of con-

tents in the fundus of the stomach closes the cardia. The discharge of chyme from the stomach is intermittent. The pylorus opens only when acid gastric contents relax the sphincter. The presence of acid chyme in the duodenum closes the pylorus and keeps it closed until the duodenal juices have rendered its contents neutral or alkaline. Acid chyme stimulates the flow of pancreatic juice and bile. The peristaltic waves press acid chyme toward the pylorus and intimately mix gastric contents and digest them. Only after duodenal contents have become neutralized can the pylorus relax and acid chyme pass through. This alternate opening and shutting of the pylorus with discharge of chyme is continued until the stomach is empty.

Gastric Secretion and Gastric Emptying are Greatly Influenced by the Kind of Food Ingested. Water and normal salt solution cause limited gastric secretion and rapid stomach emptying. This rapid exit causes but feeble peristalsis. Carbohydrate foods leave the stomach quickly because on account of their failure to unite with acid gastric juice they permit a large amount of free acid to come into contact with the pyloric sphincter and thus conduce too rapid opening of the pylorus. Marked retardation in the discharge of carbohydrates occurs if such be mixed with alkaline solutions. The alkali delays the appearance of acid by checking temporarily the secretion of acid gastric juice and also by uniting with the free acid already poured out. The acid control of the pylorus is thus interfered with and emptying is delayed. Protein food leaves the stomach slowly because proteins join with free HCl and thus for a time retard the development of an acid reaction which initiates pyloric opening. Protein has also been shown by Klingine to cause the secretion of 50 per cent. more gastric juice during the first four hours of digestion than when carbohydrate is fed. When such protein is passed into the duodenum there is more acid to neutralize than when carbohydrate was fed, consequently the pylorus remains closed for a longer time and the antrum of the stomach is subjected to intense peristaltic activity without pyloric relaxation. It is estimated that in such event the peristaltic waves number from 300 to 500 per hour. Cannon states that at the end of a half-hour eight times as much carbohydrate as protein has been absorbed, and there exists twice as much carbohydrate as protein in the jejunum. While carbohydrates begin to leave the stomach at once, proteins are delayed from one to three hours. Fats remain longest in the stomach because they excite little free HCl production and consequently the acid control of the pylorus is reduced to a minimum.

Vigorous mixing of food with acid chyme occurs in the antrum and pylorus, the zone in which 60 per cent. of all gastric ulcers are found. Food delay in this locality permits of increased local movement of the viscus, greater opportunity for food and acid to remain in intimate contact with injured gastric lining, hence, opportunity

for the maximum of trauma, infection, digestion of damaged tissue, stress on the pyloric sphincter, and local alterations in circulatory and neuromuscular mechanism.

The facts which I have enumerated have been established as constants by repeated laboratory and clinical investigation. In my opinion they furnish a logical ground-work for the treatment of peptic ulcer. They place such treatment upon a rational basis and remove from it many purely empirical and "rule-of-thumb" features which characterize commonly accepted modes of therapy. I am convinced of its clinical usefulness after five years of careful clinical observation.

5. *Selection of Cases for Types of Treatment.* The principles involved in treatment of gastric ulcer, of whatever nature, first demand *search for and eradication of the primary systemic fault.* Often the localization of such is difficult. Inasmuch as foci of infection may exist in oral adenoid tissue, head sinuses, about teeth or in systemic lymph gland chains, these must be removed promptly. Intra-abdominal infections must likewise be eradicated, *e. g.*, diseased appendix, gall-bladder, Fallopian tubes, ovaries, ulcers, or subinfections of the bowel. It would appear quite inadequate to remove external local foci of infection and to leave behind intra-abdominal foci containing bacteria already accustomed to their environment and ready to spread their operations to the gastric lining when opportunity offers. It would appear that the removal of these variously situated germ centers constitutes a fundamental step toward the cure of gastric ulcer.

After local foci of infection have been removed the mode of treatment is further influenced by the *type* of ulcer that has been proved to exist. Unless ulcers with much scar or causing great gastric deformity are demonstrated to be hectic, little hope of permanent relief by medical measures can be offered. Surgery promises the greatest prospect of relief to such cases. Intense pain, frequent hemorrhage, perforation, or the danger of malignant change taking place in callonsed ulcers likewise contra-indicate non-operative care. Unfortunately we have no clinical or laboratory tests which indicate to us *what type* of ulcers will become malignant or *when* early malignant change is taking place. The roentgen demonstration of callonsed ulcer exceeding 2 cm. diameter when such is associated with history of frequently recurring ulcer symptoms and positive chemical test for blood constantly determined in the stools, forms a clinical hint that malignancy can be expected. The most competent clinicians are agreed that callonsed, recurring ulcers, located in the pyloric end of the stomach should be treated operatively. Excision should be performed when mechanically possible. If excision is impracticable, then infolding, or cautery puncture, with or without gastrojejunostomy, yields the most satisfactory results. In non-obstructing ulcers, gastrojejunos-

tomy should always be accompanied by permanent pyloric closure. Gastrojejunostomy properly performed acts by aiding gastric emptying, by diminution of free HCl (an average of 18.2 points in 196 consecutive cases in our series), and by permitting limited jejunal regurgitation into the stomach.

Successful medical treatment of ulcer thus first demands careful selection of cases to be so treated. It is indicated only in ulcers associated with little callus, or if callonsed, located in portions of the stomach in which stenoses are not liable to result or where surgical procedures cannot be carried out. Certain essential principles are to be borne in mind in carrying out medical treatment.

II. Outline of Author's Method of Non-surgical Treatment.

1. *Rest in Bed.* Both physical and mental for from one to three weeks. Bodily and psychic activity stimulate peristalsis.

2. *Rest to the Stomach Itself.* When it is recalled that during an ordinary meal the digestive processes demand more than 2000 peristaltic waves, the effect of such as a mechanical irritant to an ulcer or the ulcer-bearing area cannot be disregarded. Complete rest for the stomach also demands avoidance of irritating medicine, gastric lavage, and frequent abdominal examinations of the suspected focus.

3. *Local Applications to the Abdomen.* Painful spasms are further prevented by having constantly applied to the abdomen compresses saturated with Ochsner's fluid (alcohol and boracic acid.)

4. *Keeping the Stomach Empty of Food.* This promotes healing by limiting local irritation from the food itself, from reducing the amount of gastric juice required to digest food, by limiting gastric peristalsis and avoiding painful gastrospasms which limit free circulatory interchange. The abstinence from food by mouth should be insisted upon for from three to seven days, according to the case. The period of fast is determined best by clinical disappearance of gastric spasm (pain, regurgitation, waterbrash, heartburn) and by fluoroscopic proof of absent or diminished gastric peristalsis. During the fast *paraffin wax is chewed* for fifteen minutes every hour. It keeps the mouth clean, promotes free flow of protective saliva and mucus, counteracts painful hunger contractions and gastrospasms, and allays thirst.

5. *Rectal Feeding.* During the fasting period, rectal feedings are instituted. From 500 to 1000 calories of nutrient mixture are given in twenty-four hours. We use a clystra containing 1 ounce of 50 per cent. alcohol, 1 ounce of glucose with normal salt solution to make 240 c.c. The nutrient enema is given at body temperature by the drop method. The drops flow at the rate of 30 to 60 drops per

minute. During the first day of rectal feeding, gtt. x of tr. opii are given with each enema.

6. *When Mouth Feeding is Begun.* Usually from the fourth to seventh day. Two factors control the choice of diet: (*a*) nourishment should be liquid and administered warm in small quantities frequently, and (*b*) carbohydrates should be selected.

(*a*) Small quantities of liquid food should be frequently administered in order that the stomach empty rapidly with the least effort and thus remain food-free for the longest time, thereby giving maximum time of rest for ulcer healing. The duodenal digestion must be called upon until gastric conditions warrant demands being made upon stomach digestion. Keeping the stomach food-free keeps hydrochloric acid or pepsin production to a minimum. From 4 to 6 ounces of warm liquid are given every hour.

(*b*) As experimental facts have established, carbohydrate foods leave the stomach most quickly. Therefore, liquid carbohydrate mixtures (barley water, rice gruel, thin cream of wheat, thin creamed vegetable soup, etc.) are fed. Milk is not given as routine. Milk results in almost pure protein clots in the stomach. These act as do other proteins and remain for a long time in the stomach as a source of irritation, as stimuli to acid secretion and as choice culture media for bacteria. If milk be given at all, it should be first parboiled or predigested. Carbohydrate liquids produce the least secretion of HCl and pepsin and are weak stimuli of gastric peristalsis and impose the minimum of work upon the duodenum. It should be recalled that the pylorus opens only when the duodenal contents are neutral or alkaline. If the gastric contents are of such nature as to impose slight demands upon the stomach secretions and motility, the duodenum has little work as a neutralizer to perform and the pylorus remains free from spasm and opens readily. There are thus avoided gastric stagnation and accumulation of distressing free and combined acids, which prevent healing and which usually demand frequent lavage or the exhibition of large quantities of alkali.

7. *Limitation of Overproduction of Gastric Acid.* This is obtained by keeping the stomach food-free as above described. This secondarily limits both the frequency and the strength of gastric peristaltic waves. Unless food leaves the stomach rapidly, gastric glands continue secretion and coincidently stress of gastric peristalsis upon the pylorus keeps up constant irritation of ulcer-bearing areas.

If the above points established by modern physiological research are borne in mind, the exhibition of large quantities of alkali are unnecessary. Their use is certainly unscientific. Providing the gastric lumen is patent, the stomach empties freely. There is no stagnant, irritating, fermenting residue. Large quantities of alkali, according to Pawlow and to our clinical and laboratory experience, create pernicious increases of gastric acid and of mucus and generally

demand relief by lavage. Moreover, we have shown that many gastric ulcer cases do not exhibit hyperacidity or hypersecretion. It is true that the stomach *can* neutralize large quantities of alkali if compelled to do so, but there is no physiological reason why it should be called upon to thus overwork. It will be remembered that the normal habitat of gastric epithelium is an acid or at the best a neutral medium. If these epithelial cells are called upon to live in an excess of alkali, they live, as it were, in the presence of a foreign body. Experiments in artificial tissue growth have shown that cell proliferation is retarded by hyperisotonic alkaline solutions. Hence, overalkalinization may prevent healing. Attempts at protection from this foreign body (excess alkali) are shown (1) by the acid-producing glands oversecreting, and (2) by the mucoid degeneration of physiological fatigue which results in the throwing out over the secretory glands of a protective layer of mucus. The vicious circle thus formed results in enormous secretion of acid and mucus and is doubtless at least a partial explanation of the so-called hypersecretion associated with gastric ulcers, particularly when such are treated by the overalkalinization method. To combat this condition of affairs the patient's stomach must be washed frequently or greater quantities of alkali must be given in order to overpower the stomach's defensive mechanism and produce fatigue or exhaustion of the acid-secreting mechanism. It is a common observation that those patients who are treated for ulcer by the overalkalinization procedure always require frequent gastric lavage in order to ensure their comfort. This frequent lavage is to be condemned not only on account of its disagreeable features but because it acts contrary to the primary requirement of healing, namely, *rest of the affected part*. It is quite evident to those who have watched the behavior of a stomach by means of fluoroscopic screen when a tube is inserted into it that gastric lavage defeats this primary principle of healing. Lavage is generally accompanied by vigorous gastric contractions that persist not only during the maneuver but often for a long time afterward. If dieting is arranged on the carbohydrate basis, alkali is given in only sufficient quantities to keep the stomach slightly acid or neutral and to neutralize the duodenum, thus aiding in pyloric relaxation, gastric lavage need rarely be instituted during the entire course of a patient's treatment. In the past five years we have not employed lavage therapeutically in ulcer cases more than a dozen times. Lavage is so rare a procedure in my clinic that my associates and patients consider such an order as almost contra-indicated. It is readily judged how a treatment of which lavage does not form a prominent feature contributes much to a patient's peace of mind and shortens the period of hospital incarceration.

8. *Medical Treatment.* It is doubtful if any form of medicine has a direct healing effect upon peptic ulcer. Medicines are adminis-

tered largely to counteract discomfort due to three main causes, namely, (*a*) painful gastrospasms, (*b*) accumulations of overacid gastric contents associated with peristaltic unrest, (*c*) pain associated with perforation.

(*a*) *Painful gastrospasms* are usually controlled by carrying out the dietetic principles which I have above mentioned. The chewing of paraffin wax relaxes the pylorie spasm largely through stimulating a proper swallowing reflex and by fatigue of hunger-like contractions. Certain types of case in which there is an individual vagus hypertonia, or when ulcers are located at or near the orifices, demand the exhibition of antispasmodic medicines, such as atropin, tincture of belladonna, or bromides. In the early stages of the treatment, when the stomach is being kept as free as possible of contents, atropin may be given hypodermically or bromides may be placed in the nutrient enemata. Later, when food is being given by mouth, tincture of belladonna in doses of from 5 to 15 drops may be administered fifteen minutes before feeding from three to six times daily. We have not found useful, as analgesics, the exhibition of large doses of such "protective" medicines as bismuth and olive oil. These medicines doubtless act by affecting the rate and intensity of peristalsis, although they may have some effect in proved cases by direct action upon the ulcer. At times orthoform, given in 10-grain doses, in warm water, is an efficient local anesthetic when it is able to come in direct contact with an open ulcer.

(*b*) *For the relief of overacid gastric accumulations*, sodium bicarbonate is contra-indicated, because its administration results in the production of annoying accumulations of carbon dioxide with resultant gastric retention or painful belching, and because its neutralizing value is comparatively low. Large quantities of bicarbonate of soda are necessary to give relief, and the administration of such secondarily produces excessive gastric secretion. If alkalies are indicated, better results are obtained by the exhibition of frequent small doses of milk of magnesia or calcined magnesia. The ordinary case is very comfortable when from five to ten grains of calcined magnesia are given every two or three hours. Many cases require no exhibition of alkali if the physiological principles above outlined form the basis of the treatment. Only in very extreme cases is it necessary to employ gastric lavage. When it is employed, warm Carlsbad water (1 dram of artificial Carlsbad salts to 1 quart of water) may be satisfactorily administered. Usually the exhibition of atropin or belladonna for the relief of gastrospasms exerts a definite effect toward controlling oversecretion of acid gastric juice.

(*b*) The acute prostrating *pains of perforation* are best controlled by the prompt administration of morphin hypodermically, rest in bed, and hot compresses to the abdomen. Only prompt surgery saves the patient's life.

9. *Hemorrhage.* Constant seepage, demonstrated either microscopically or clinically, is generally an indication for abdominal section. Intermittent seepage may be controlled best by rest in bed, morphin hypodermically, intravenous injections of fresh horse serum, coagulose, or by copious transfusion of whole blood. In acute hemorrhage accompanied by vomiting, prompt lavage of the stomach with water at 110° F. frequently stops both the vomiting and the hemorrhage. The exhibition of morphin, rest in bed, and whole blood transfusions generally prevent recurrence of hemorrhage. In this class of case, however, surgical intervention should be resorted to early, and this especially if in a given patient, frequent, copious, prostrating hemorrhages occur.

10. *Bowels.* During the early periods of treatment, simple soapsuds enemata may be administered every second day. After the second week, morning doses of phosphate of soda or Carlsbad salts in hot water may be applied. In chronic cases, liquid paraffin given in equal quantities of warm cream result in easy motions, and the paraffin appears to have certain protective value upon the ulcer bearing area.

Summary of the Author's Dietetic Régime in Treatment of Peptic Ulcer.

Days 1 *to* 7 (Time varies as outlined in above description of treatment).

By Mouth. One-half ounce warm water hourly when awake. Patient chews paraffin wax for fifteen minutes at least once in two hours. Juice of sweet orange or grape fruit occasionally.

By Rectum. Nutrient enema consisting of 50 per cent. alcohol 1 ounce, glucose syrup 1 ounce, and normal salt solution 6 ounces every four hours. The enemata are preceded by a cleansing irrigation of the colon with normal salt solution. They are given at body temperature by the drop method at the rate of from 30 to 60 drops per minute. Calories daily approximately 1000. During the first two days, tr. opii ℥x is added to each second enema.

Days 3 *to* 14 (case of average severity).

By Mouth. From 4 to 6 ounces of water gruel at temperature of 100° F. The gruel is taken slowly through a glass tube. Gruels are made from rice, cream of wheat, oatmeal, sago, corn-meal, malted milk, macaroni and vermicelli, rusks, potato, asparagus, cauliflower, beans, peas, and boiled onion. They are strained before feeding. Flavoring with coffee, chocolate, vanilla, caramel, etc., renders the cereal gruels palatable and their administration easier. To the vegetable gruels small quantities of arrowroot or corn-starch are added to secure a thin emulsion.

Before each feeding, paraffin wax is chewed for five minutes. Warm water or sweet orange or grapefruit juice are allowed as desired, but never in greater quantity than 1 ounce at a time.

By Rectum. During the first two days of mouth feeding, two alcohol-glucose-saline nutrient enemata are given. During the second two days, *one* such nutrient enema is administered. After the fourth day of mouth feeding no rectal feedings are given in the average case. Calories approximately 800.

Days 14 to 21.

6.30 A.M. A glass of hot water and 1 teaspoonful of non-effervescent sodium phosphate.

7.30 A.M. One ounce of sweet orange or grapefruit juice, 2 ounces of thin cream of wheat, or farina, or well-cooked rice, or corn-meal, 2 ounces of skimmed, parboiled milk, may be taken with cereal, and if desired a small quantity of powdered sugar used; 1 zwieback with a thin layer of fresh butter, 4 ounces of parboiled skimmed milk, containing half volume of lime-water, served warm and flavored with coffee, cocoa, caramel, or vanilla.

9.30 A.M. Six ounces of thin water gruel from cereals or fresh vegetables, strained and served hot, 1 rusk or zwieback, or dry toast.

11.30 A.M. Four ounces of malted milk, whipped egg, with parboiled milk, corn-starch pudding, simple custard lightly cooked.

12.30 P.M. Six ounces potato, pea, bean, or asparagus puree, (strained), or vegetable broth; 4 ounces of salisbury steak (moderately well cooked) to chew; 2 ounces (cooked weight) of thin rice, sago, tapioca, or corn-starch pudding made with parboiled milk and egg yolk; 2 ounces of parboiled milk and small quantity of polverized sugar may be eaten with the pudding; 1 rusk or zwieback, 6 ounces of parboiled milk and quarter volume of lime-water flavored to taste.

4 P.M. Four ounces of water gruel from cereals, 1 very soft poached egg, 1 rusk or zwieback, 4 ounces of hot Vichy water.

6 P.M. Four ounces of whipped egg, 2 rusks or zwieback, 6 ounces of malted milk (thin), flavored to taste, or cereal water gruel or parboiled milk and quarter volume of lime-water gruel.

9 P.M. Six ounces of water cereal gruel or 4 ounces of malt marrow, 2 graham crackers. Calories approximately 1500.

Days 21 to 42.

6.30 A.M. Two teaspoonfuls of phosphate of soda in a glass of hot water.

8 A.M. Juice of 1 sweet orange or half sweet grapefruit, or boiled prunes passed through fine colander; 2 ounces (cooked weight) of thin cereals (cream of wheat, farina, oatmeal, corn-meal) 2 ounces of skimmed milk and small amount powdered sugar, 1 soft poached egg, 2 zwieback, 2 rusks or 2 thin slices of well-toasted graham bread, 1 pint of hot skimmed milk + a quarter volume of lime-water flavored to taste (cocoa, vanilla, etc.).

10 A.M. One pint of hot parboiled whole milk and fifth volume of lime-water, 2 rusks or graham crackers.

12.30 noon. Four ounces of creamed soup from vegetables, strained, 6 ounces rare meat to chew. 4 ounces well-mashed potato or baked potato (mealy inside), or carrot, peas, beans, cauliflower, Brussels sprouts, or asparagus (all vegetables passed through a strainer and served with 15 grams of butter), 4 ounces (cooked weight) of pudding from rice, corn-starch, sago, tapioca, cream of wheat, or farina or 4 ounces of custard, pulp of sweet orange, grapefruit, or prune whip, or chew 6 ounces of watermelon or cantaloup, half pint of hot skimmed milk.

3.30 P.M. One hundred and fifty c.c. of hot whole milk and quarter volume of lime-water or 150 c.c. of malted milk or weak cocoa.

6.30 P.M. Two rusks or zwieback or 2 slices of well-toasted graham bread, 2 very soft poached eggs, 100 grams of sweet apple sauce or 1 baked apple (omit skins), or juice of sweet orange or half of grapefruit, or chew 6 ounces of melon, 1 pint of skimmed milk, hot.

9 P.M. Two hundred and fifty c.c. of whole parboiled milk and quarter volume of lime-water of 250 c.c. of malted milk, hot. Calories for twenty-four hours approximately 2000.

General Diet after Three Months. If distress, patient should go back to 7 to 21 day diet.

7 A.M. One pint of skimmed milk and half-gill of cream.

9 A.M. Two pieces of toast without butter, juice of one sweet orange or grapefruit or ripe melon or apple sauce or baked apple (do not eat skin) or marmalade, 1 dish or well-cooked cereal (oatmeal, farina, or cream of wheat), 2 very soft poached eggs, 2 cups of hot, sweetened water. The water may be made more palatable by flavoring with cocoa, tea, coffee, or cream.

11 A.M. One cup of bouillon (two cubes), two graham crackers.

1 P.M. This should be the heavy meal of the day. It may consist of meat (rare beef, rare hamburger steak, lamb, or white meat of fowl), fish (never fried), oysters, well-cooked spinach, cauliflower, carrots, squash, peas, (milled), string beans, Brussels sprouts, baked or mashed potatoes (in moderation), rice with gravy, simple puddings made from cereals, corn-starch, gelatine, well-cooked fruit sauces, simple cakes, no white bread (all bread should be made from dark flour and should be at least one day old), 1 pint of skimmed milk taken hot.

4 P.M. One glass of hot peppermint water (20 drops of "essence" of peppermint to the glass), sweeten to taste and drink slowly; two graham crackers.

7 P.M. A light lunch consisting of vegetable soup, simple salad, toast, soft eggs, and plain puddings or cake, with or without ripe cooked fruit sauces; 1 pint of hot skimmed milk.

Bedtime. One glass of malt marrow, malted milk, or hot skimmed milk. Calories approximately 3500 for twenty-four hours.